

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

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| Application No.: | 10/516,705 | Art Unit: | 1643 |
| Filed: | December 2, 2004 | Examiner: | L. Bristol |
| 1 st Inventor: | T. Hara | Allowed: | |
| For: | Mutant Androgen Receptor, Cancer Cells Expressing the Same, A Method of Producing Them and Use Thereof | Batch: | |
| Atty. Dkt. No. | 3056 USOP | Paper No.: | |

Election of Claims

Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

Dear Sir:

In response to the Restriction Requirement mailed January 12, 2007 for the above-identified U.S. patent application, Applicants hereby elect to prosecute the aspects of the invention set forth in Group V (claim 12) with traverse. No amendment of inventorship is necessitated by this election. A Petition for a One-Month Extension of Time and an authorization to pay the 37 CFR Sec. 1.17(a)(1) fee of \$120.00 accompanies this response.

In response to the Examiner's comments, Applicants provide the following information for consideration.

One of the characteristics of the present invention is production of an anti-androgen drug-resistant cancer cell line that expresses a mutant androgen receptor, by culturing (i.e. *in vitro*) cancer cells sensitive to a specified anti-androgen drug in the presence of said anti-androgen drug. This characteristic is quite different from introduction of a mutation into HeLa cells using pARL plasmid containing a mutated AR sequence derived from the LNCaP cell line described in Veldscholte *et al*, which was established from tumor cells derived from a metastatic lesion of a human carcinoma, and which expresses a mutated androgen receptor (AR).

As to the Hara *et al*. reference, Applicants note that their earliest priority Japanese patent application was filed on June 3, 2002, prior to the publication of the article.

As concerns the Culig *et al.* reference, the LNCaP-abl cell line described therein was obtained by culturing LNCaP cells in androgen-depleted medium for a long period, and therefore a novel mutation is not introduced into AR gene (protein) in the cell line.

Furr *et al.* only describes that flutamide enhances the proliferation of LNCaP cell line. As understood from above, the flutamide resistance of the cell line did not result from introducing a mutation into AR *in vitro*.

The references cited by the Examiner do not disclose or suggest the technical feature of the invention as set forth in independent claim 1, wherein a mutation can be introduced into AR *in vitro* by culturing cancer cells sensitive to an anti-androgen drug in the presence of said anti-androgen drug. For this reason, Applicants disagree that the technical feature recited in claim 1 is not special.

Early allowance of the claims is requested. Should the Examiner believe that a conference with Applicants' attorney would advance prosecution of this application, the Examiner is respectfully invited to call Applicants' attorney at the number below.

Respectfully submitted,

Date: March 9, 2007

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